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Culture as a Stressor: A Revised Model of Biocultural Interaction

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ABSTRACT Contemporary urban societies display in high relief the action of social stratification on human biology. Recent studies of biological responses to urban environments and of socioeconomically disadvantaged people indicate that culture allocates risks disproportionately to some individuals and groups within society through its constituent values and related patterns of behavior. Although risk allocation is present in all societies, it is very clear in urban environments within stratified societies where high exposure to harmful materials is many times more likely for some segments of society. In urban environments, culture may be seen as adding stressors to the environment by concentrating naturally occurring materials to levels that are toxic to humans and through the creation of new toxic materials. In stratified societies the risk of exposure to these new stressors is focused on the socioeconomically disadvantaged. This exposure has consequences that increase the likelihood of more exposure and more socioeconomic disadvantage, thereby increasing social stratification. This suggests that models of biocultural interaction include a feedback relationship in which biological factors influence the sociocultural system in addition to the usual action of the sociocultural system on biological features and responses. This model strongly reinforces the view that stressors can originate from cultural arrangements. Am J Phys Anthropol 102:67–77, 1997

Concepts of culture and models of biocultural interaction have changed substantially over the past 20 years (Dressler, 1995) and have been the subject of much discussion and debate (Goodman et al., 1988; Singer, 1989; Wiley, 1992). The study of people's biological reactions to features of urban environments exposes an action of culture not emphasized previously but an action that is consistent with interpretations of culture current in contemporary ethnology. Ethnologists now see culture as less coherent, less problem-solving than had been conceptualized previously, and as not shared by all or the same for all members of society (Rosaldo, 1989; Gossen, 1993). One conclusion from studies of the human biology of urban environments is that technological innovations can create stressors which entail subsequent adjustments or adaptations. However, these stressors are not experienced equally by all social groups.

The view that culture can introduce stressors rather than buffer them is consistent with observations on the role of social factors in the distribution of infectious disease (Fenner, 1970; May, 1960) and with the observation that culture, through political and economic factors, may constrain the choices that people can make in responding to their environment (Armelagos et al., 1992). This paper suggests that political and economic factors not only constrain choices but also inequitably distribute human-made stressors within urban populations. It describes some specific modes by which culture

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can introduce stressors and shows how culture can focus a stressor on socially defined groups, which ultimately affects culture itself. Thus, this essay presents a model of how biological response can influence social structures.

CULTURE AS A STRESSOR: SOME MODES OF ACTION

Culture can act to present new biological challenges to humans in a myriad of ways. Based on observations in contemporary urban environments in industrialized countries and from the study of their history, the following four modes of action may be distinguished.

1. The invention of new materials such as PCBs (polychlorinated biphenyls), PBBs (polybromated biphenyls), and DDT (dichlorodiphenyltrichloroethane), for example, can add stressors to the environment. Human adaptation has evolved human biological systems to fit a range of environments and their components. Completely novel materials in an environment are more likely to pose a challenge to survival and adaptation than materials that have been part of the environment for much of human existence and to which humans have evolved. Culture is not the only originator of materials unfamiliar in human evolutionary history. Natural forces create new compounds, but culture, through technology, has been a noticeable contributor of new materials.
2. A second mode is the concentration of naturally occurring materials that are not damaging or stressful at low doses but, once concentrated, become so. Numerous examples exist: lead, nickel, mercury, and energies such as noise and radiation are abundant in the environment at low levels. When concentrated, each interferes with some aspect of human functioning and survival (Schell, 1991). Noise, for example, when experienced at most naturally occurring levels, does not result in immediate hearing loss or presbycusis, but concentrated noise exposure such as is often found in cities of industrialized societies has made hearing loss commonplace.

   Human activity is not the only means of concentrating materials and energy. Nonhuman animals concentrate materials. For example, shellfish concentrate mercury, but human activity has been the major force responsible for the concentration of many materials to levels toxic to humans.
3. Culture also reschedules the timing of exposures to infectious agents as well as to naturally occurring materials and energy from a time when adaptive responses are possible to one when such adaptive responses are less likely to occur. There are several examples from the natural history of infectious disease. In human, mumps produces quite different and more serious symptoms and sequelae when contracted after childhood (Kiple, 1993). Poliomyelitis most always produces a subclinical infection when exposure is very early in postnatal life, but when infection occurs in adolescence it is more likely to spread from the gastrointestinal tract to the central nervous system (CNS) and is more likely to cause paralysis (Burnet and White, 1972). During the sanitary revolution in Europe, the nations that had reduced exposure to infectious agents, especially those transmitted by the oral-fecal route and waterborne ones, also postponed exposure to the poliomyelitis virus until childhood or adolescence. This increased the risk that CNS symptoms would follow from infection. In fact, paralytic polio was epidemic first in the most sanitized nations of northern and western Europe in the nineteenth century (Burnet and White, 1972). The relationship between sanitation and poliomyelitis infection can be seen in a cross-sectional view as well by comparing the ages at which 50% of the populations have serological evidence of past infection. In 1952, antibodies to type 2 poliomyelitis virus were present in 50% of the population of Cairo by the early age of 14 months, but in urban areas of the US antibodies were not present in 50% of the population until the age of 10–15 years (Paul et al., 1952, cited in Fenner, 1970).
4. The fourth mode is the allocation of risk to some segments of society more than to

others. Risk allocation can be described in terms of risk factors—that is, a characteristic of the individual that changes the probability of occurrence of a specific disease or any biological outcome (for several definitions see Last, 1988). Risk factors may be genetic, behavioral, residential, or occupational. Alleles and genotypes are risk factors for specific diseases (e.g., blood type O is a risk factor for duodenal ulcers [Polednak, 1989]). Cigarette smoking, a behavior, is a risk factor for lung cancer and heart disease (US Department of Health, Education, and Welfare, 1979). Residential proximity to a swamp infested with malaria-carrying mosquitos is a risk factor. Many occupations are risk factors because they involve exposure to materials that produce disability, disease, and/or death.

Risk factors that are associated with groups defined by social ascription rather than by biological characteristics may be predominant in stratified, industrial societies. For example, the National Coalition of Hispanic Health and Human Services Organizations examined data from the Environmental Protection Agency (EPA) relating to environmental justice (National Coalition of Hispanic Health and Human Services Organization, 1994). US Hispanics are far more likely to live in areas failing to meet EPA air-quality standards than non-Hispanic blacks or non-Hispanic whites (Wernette and Nieves, 1992). They are more than twice as likely as whites to live in areas with elevated levels of particulate matter and twice as likely as non-Hispanic blacks (Wernette and Nieves, 1992). They are far more likely to live where lead and carbon-monoxide levels are elevated (Wernette and Nieves, 1992). Since most of these exposures are related to area of residence, residential segregation by ethnicity clearly is a factor in understanding urban human biology. The other main organizer of risk for US Hispanics is occupation. Most seasonal agricultural workers are Hispanic (Mines, 1991) and as such are exposed to higher levels of organophosphate and organochlorides than other segments of society. In short, biological characteristics and behavioral regularities produce risk.

In terms of a model of biocultural interrelation, untoward biological outcomes are distributed within society through the operation of risk factors, many of which are social characteristics of individuals and of groups (Schell, 1992). The social relations through which some segments of society exert power over others structure this differential allocation of resources and risk. These inequalities are reinforced by creating social distinctions that become the basis for patterns of behaviors that determine exposure to materials that can produce untoward biological outcomes.

In some societies sex and age may allocate risk—for example, in the additional risk to health associated with childbearing. Biological features such as these are not culturally allocated in the strictest sense. Yet it is more often the case that age and sex are used to make important cultural or social distinctions that serve to structure behavior that may carry risk and can be the basis of social and economic discrimination.

By studying highly specialized societies, many means of risk allocation can be discovered. In societies with social stratification, occupational roles, and associations, the risks to health and the impacts on human biology are allocated very specifically. However, less specialized societies are not free of risk allocation. Shamans may experience extra risk because of their behavior which may include vision quests, or social isolation. Another example of risk allocation comes from highland New Guinea where the risk of contracting Kuru was closely related to the practice of prescribed funerary rituals. Thus, insofar as some activities are performed by some people more often than others, there can be additional risks to that activity that pertain to human biology in any society.

In addition to the four modes of action delineated above, there usually is interaction among them. The most common is the

interaction of risk allocation with one or more of the other three modes. Examples are evident in industrial cities because of the range and concentration of toxic materials in cities of industrialized societies and the severe social stratification that often exists there. Once natural materials are concentrated to toxic levels or new toxic materials are introduced, a significant exposure can occur in an occupational or residential setting, both of which are socially structured.

Similar interactions involving infectious agents are well known. For example, exposure to infectious agents may be rescheduled for some socially defined groups but not others, with important consequences. The change in the distribution of poliomyelitis during the first half of the twentieth century provides an example of the interaction of rescheduling an exposure and risk allocation. As the US and European countries improved sanitary conditions during the first half of the twentieth century, the likelihood of exposure to the poliomyelitis virus in infancy was reduced. Since sanitary improvements occurred earlier in wealthy areas, the reduction in exposure was greatest among the wealthy. Since postponing exposure to the poliomyelitis virus from infancy to adolescence increases the likelihood of paralytic sequelae, the rates of paralytic polio were higher among the wealthy than the poor (Burnet and White, 1972) until vaccines were introduced. Thus, social stratification and the power relations that it involves influence occupation and residence which are literally instrumental in allocating risk of exposure to toxic and infectious materials.

THE INTERACTION OF CULTURAL STRESSORS: RISK FOCUSING

Risk focusing refers to the sociocultural process of allocating exposure to toxic or infectious materials to groups whose members enter the group partly because of previous exposure to those materials (Schell, 1992). This is most easily referenced on an individual level. A severe toxic exposure by individuals may produce disability, disqualify them from their occupation, and result in downward social mobility. Occupational choices become limited to less rewarding jobs, socioeconomic status may become lower, and housing away from residential pollution may be unaffordable. Such occupations and residences may lead to increased exposure. In short, exposure to toxic materials can lead to increased exposure, or low social status can lead to lower social status through the effects of toxic exposure(s).

Figure 1 illustrates the process on the group and individual level. Occupations and residences available to individuals of low socioeconomic status lead to greater risk of toxic exposure, leading to toxic insult which can lead to disability. This in turn results in poorer qualifications for employment leading to lower occupational status which results in lower socioeconomic status. On the other hand, individuals of higher socioeconomic status have occupations and residences with less exposure to toxic materials, thereby preserving them from insult and disability. This in turn results in better qualifications for employment and higher paying occupations with less toxic exposure. Choices of residence are greater and more likely to be away from environmental sources of toxic exposure and insult.

While the risk-focusing process may unfold in one generation, it may also occur over generations. If exposure to toxic materials occurs during development, especially fetal development, functional cognitive behavioral deficits may result. Substances that impair postnatal cognitive and behavioral development following prenatal exposure are termed behavioral teratogens. There are several known, but alcohol is the best known. Heavy alcohol consumption during pregnancy can produce fetal alcohol syndrome in the offspring, a syndrome of structural alteration in the cerebral cortex, poor cognitive functioning, distinctive facial dysmorphology, postnatal growth impairment, and other effects (Clarren and Smith, 1978). Mercury is a behavioral teratogen as well, and lead may be one too. Prenatal mercury exposure produces in the brain recognizable lesions which are related to behavior (Waldbott, 1978). Evidence of lesions or structural alterations from prenatal lead exposure is not conclusive even though there is strong evidence for behavioral alterations.

Exposure to materials associated with impaired postnatal development, whether they

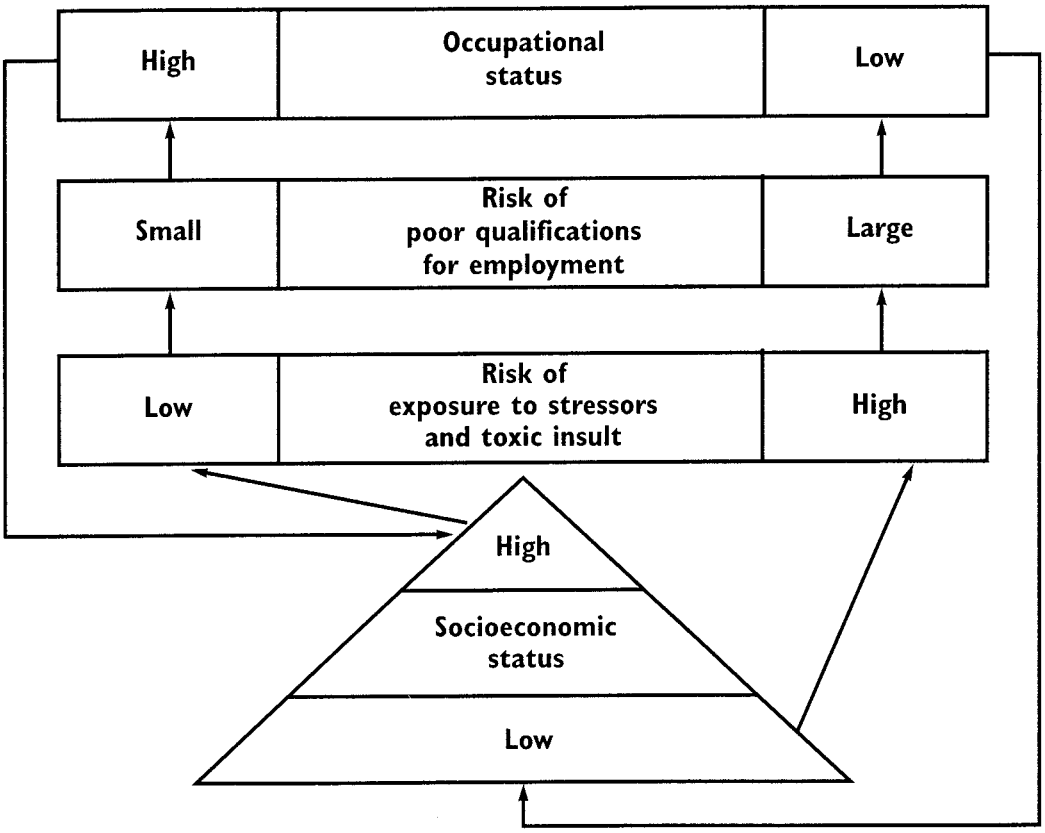

Fig. 1. Generalized risk focusing model (Schell, 1992).

formally qualify as behavioral teratogens or not, may be part of a common cycle of risk focusing that unfolds across generations in industrialized cities. The result of risk allocation and risk focusing can be seen by examining the distribution of lead and its effects in the US.

Lead is dispersed at trace-level concentration in the natural environment, but a variety of human activities concentrate lead so that an acute exposure can cause severe effects on growth, cognition, and many other physical parameters (Damstra, 1977). Chronic exposure to ambient lead in air, food, and water also produces detrimental effects on growth, cognition, and physiological parameters (Centers for Disease Control, 1991; Davis and Svendsgaard, 1987; for review see Needleman, 1992). Children are at greater risk for lead toxicity compared to adults because children are exposed to more lead dust through play activities, and once exposed they absorb a far greater percentage of ingested lead from the gastrointestinal tract than adults (Annest et al., 1982). Also, children absorb more lead through respiration (their respiratory rate is higher, and their exchange rate per unit body mass is higher) than adults (Annest et al., 1982; McCabe, 1979).

In the US population, lead levels vary considerably according to culturally mediated forces such as race, social class, and place of residence (Annest et al., 1982; McHaffey et al., 1982). Data from the NHANES II survey conducted from 1976–1980 show that, among children between 6 months and 5 years of age, those living in cities of a million or more inhabitants had a greater frequency of elevated lead levels (defined as 30 μg/dL). Low income and nonwhite ethnicity were also associated with

elevated lead levels. Differences in exposure by race and residence were extreme. Only one in 50 white children between 6 months and 5 years of age had lead levels elevated to 30 µg/dL or more, but one of six black American children of the same age living in the central areas of large cities had an elevated lead level, more than an eightfold increase in risk of elevated lead level. Data from the Third National Health and Nutrition Examination Survey conducted from 1988–1991 show that lead levels have decreased substantially in the US (Pirkle et al., 1994). At the same time, however, studies conducted since NHANES II show that lead produces detrimental effects at levels far below 30 µg/dL, and the CDC has set the action level at 10 µg/dL (Centers for Disease Control, 1991). The data from NHANES III (Brody et al., 1994) show that among non-Hispanic, black American children from 1–5 years of age who live in the central areas of cities of 1 million or more inhabitants, 36.7% have lead levels above the action level. In contrast, only 6.1% of non-Hispanic, white children in the same environment have elevated lead levels, and 5.2% of non-Hispanic, white children in noncity environments have elevated levels. The distribution of elevated lead burdens among US children is not the result of recent environmental exposure but of a multigenerational experience with lead in the environment. Lead is transferred from mother to fetus during gestation (Alexander et al., 1973). Today infants are born with a lead burden acquired transplacentally and attributable to their mother's exposure.

The risk-focusing model as it applies to lead is depicted in Figure 2. It begins with residence during childhood in a neighborhood where ambient lead levels in the environment are high. Such neighborhoods have been common in many older cities where lead dust from standing, dilapidated, lead-painted homes, demolished buildings, and automobile exhaust are present in the air and concentrated in the soil (Duggan and Inskip, 1985; Lin-Fu, 1979). Childhood residence in such neighborhoods results in high lead exposure and absorption. A high blood-lead level has many affects on the individual, most importantly cognitive and behavioral deficits (Davis and Svendsgaard, 1987). These deficits may increase the chances of doing poorly in school and of leaving school before completing secondary school (Needleman, 1992). Poor school performance may contribute to poor preparation for employment, early fertility, and other outcomes. A fourth factor, poor knowledge of dietary requirements, does not result directly from not completing school, but it would be one of several outcomes related to suboptimal education that would be more likely. Poor knowledge of dietary requirements may result in a suboptimal diet during pregnancy and suboptimal household nutrition once children are born. Poor diet during pregnancy may increase the release of maternal lead stored in bone during pregnancy (Silbergeld, 1986), while a mineral-poor and fat-rich diet during childhood may increase the risk of lead absorption (Barltrop 1969, 1982). Among people already at risk for lead exposure, such diets may be more common.

In this model, the parents' poor preparation for employment increases the risk of lead exposure for the next generation since poor job preparation increases the risk of obtaining a lower paying job or being unemployed. Both can lead to limited choices for residence and an increased chance of residence in an older neighborhood where rents are lower (i.e., in an older neighborhood where environmental lead levels are high).

When a poorly educated young adult from a neighborhood with high ambient lead levels becomes pregnant, it is likely she will not receive early prenatal care, including dietary counseling. With a mineral-poor diet, she may transfer lead across the placenta to the fetus and deliver a child with a higher lead burden at birth. When a postnatal diet that promotes the absorption of lead from the environment and residence in an environment with high ambient lead are added, the next generation may acquire a considerable lead burden of its own before entering school. With a lead burden above 10 µg/dL acquired in time for school, the cycle of cognitive/behavioral deficits, difficult poor school performance, and early school leaving may begin again.

This interaction illustrates several of the modes by which culture is a stressor. A naturally occurring material, lead, is concen-

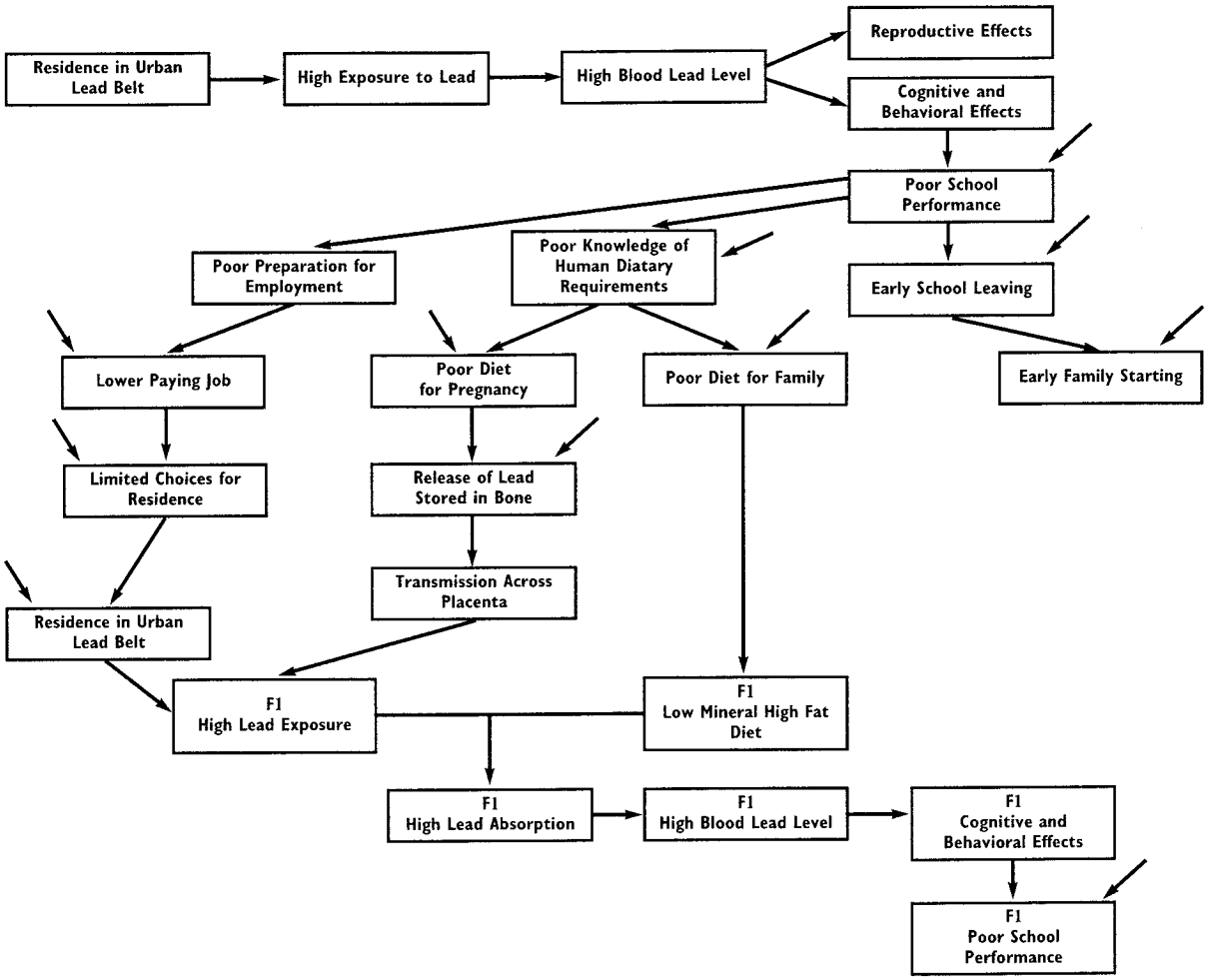

Fig. 2. Risk focusing model as applied to lead exposure and effects (adapted from Schell, 1992). An arrow that does not originate from a box indicates the existence of other influences that could not be depicted in the diagram.

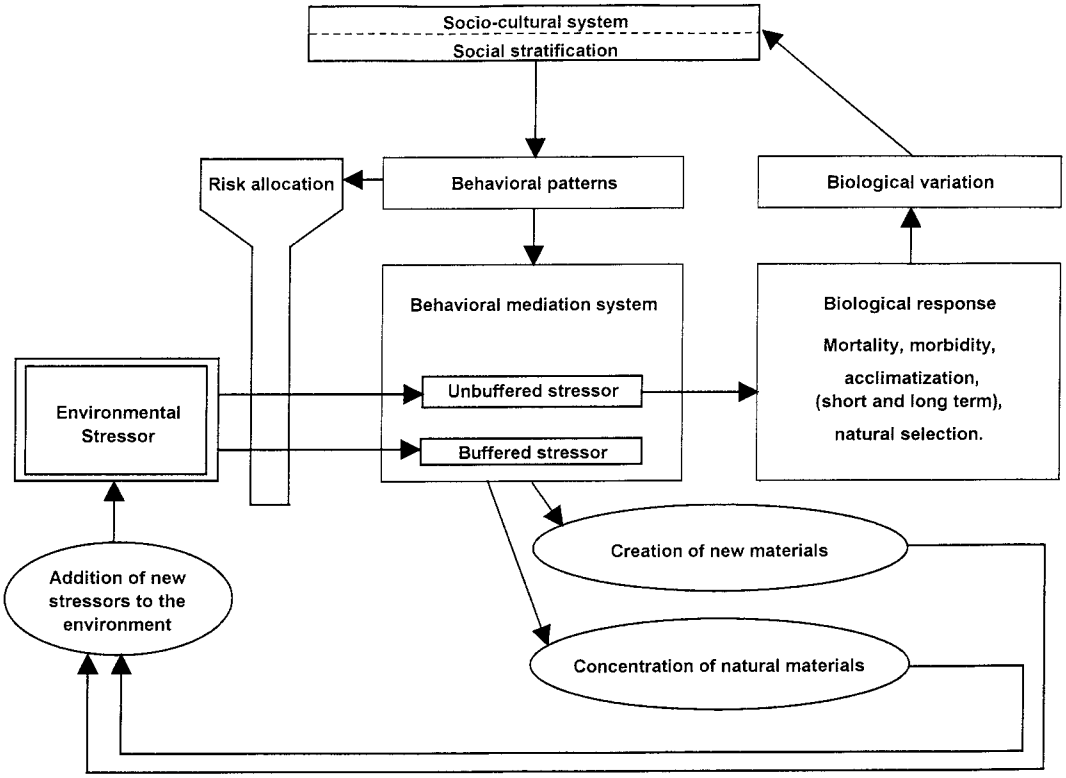

Fig. 3. Model of biocultural interaction revised to incorporate risk allocation, risk focusing, and the introduction of environmental stressors by the sociocultural (behavioral buffering) system.

trated to toxic levels by numerous current and past industrial activities. Exposure to the material is strongly influenced by membership in socially defined groups such that social attributes are risk factors for extra exposure to lead. When heavy exposure is rescheduled to the fetal period or to childhood, a permanent cognitive impairment can result. Over time, exposure to lead can result in a social trajectory that is likely to result in additional exposure to lead. Exposure has become focused on some socially defined groups.

IMPLICATION FOR MODELS OF BIOCULTURAL INTERACTION

Risk focusing is an example of a positive feedback system. If uninterrupted, the cycle of exposure, impairment leading to more exposure, and more impairment continues. As it does, social stratification also increases. Thus, risk focusing can exacerbate social inequality and contribute to stratification within society.

Figure 3 presents a graphic version of some features of a model of biocultural interaction (not all features can be displayed graphically—e.g., rescheduling). The illustration emphasizes the role played by behaviors that attempt to buffer stressors, termed *behavioral mediation* (following Thomas, 1975), in adding new stressors to the environment. The lower portion of the model depicts effects of the behavioral mediation system in creating stressful new materials and concentrating naturally occurring ones to stressful levels. This can occur when technological adaptations produce pollutants. The top portion of the model recognizes the role of biological variation as an input into the sociocultural system. Social systems are influenced by biological variability because sociocultural systems often incorporate biological variation into their ideational sys-

tems and because biological variation may contribute to social stratification through risk focusing. Some biological differences can result from differential pollutant exposure, and such differences can damage the individual's ability to secure economic advantages in society and promote downward socioeconomic mobility. The result is the downward spiral depicted earlier in Figure 1. The model also emphasizes the role of behavioral patterns that are part of the behavioral mediation system in allocating risk to specific social groups. Risk allocation is interposed between the environmental stressors (which are not necessarily aspects of the physical environment) and the behavioral mediation system. It should be noted that risk is allocated by a whole complex of behaviors that are usually associated with social stratification and not necessarily by the specific stress-buffering behaviors. The notion that culture reschedules stressors could not be displayed as a separate pathway in the figure, but it can be considered a form of risk allocation.

Although consistent with previously described models of biocultural interaction, the model has a few distinctive features: the influence between biology and culture is bidirectional. It is proposed that risk focusing is a mechanism by which biological factors can influence culture through their impact on the social configuration, specifically the degree of social stratification. Biological difference is not the cause of social stratification, but it may become a contributor to the latter. Previous conceptions of the relation between biology and culture are based on one of two models of the interaction. In the earlier model there is a clear separation between the two realms: cultural variation is independent of biological factors and vice versa. In this antiquated model, once culture evolved in the human record, human evolution was supplanted by cultural evolution, and biological change ceased or at least became insignificant. Contemporary microevolution is not incorporated in this model. The term *biological substrate* evokes the older model because it implies that biology only underlies human culture rather than interacts with it. In the second model there is a relationship between biology and culture, but it is unidirectional; culture affects biology. For example, consider how the relationship between slash-and-burn agriculture, mosquito ecology, and the distribution of malaria in sub-Saharan Africa has been described. Although Livingstone (1958) and Weisenfeld (1971) showed that the expansion in sub-Saharan Africa of wet agriculture was increased by populations with a high frequency of the sickle cell gene, which is an example of biological variation having an impact on culture (subsistence strategy), most anthropology texts focus on the role of agriculture in intensifying humans' exposure to the mosquito vector, which is an example of culture's impact on biological variation. In many presentations of biocultural interactions, the impacts of biological variation on culture are often overlooked in favor of a model in which culture affects biology. (While it should be remembered that introductory physical anthropology texts do not necessarily have to include a discussion of sickle cell and agriculture as a biocultural interaction, it is interesting to compare the different approaches among popular texts such as Molnar (1992), McElroy and Townsend (1989), Weiss and Mann (1990), Bennett (1979), and Relethford [1990]).

There are other examples of biological variation affecting social stratification, subsistence, and other aspects of culture. Greene (1974) described a biocultural interaction in highland Ecuador. Low socioeconomic status increased the risk of ingesting certain foods during times of economic hardship. Some of these foods were probably goitrogenic. The ingestion of goitrogenic foods combined with a diet chronically low in iodine may have lead to disturbed cognitive development and a high frequency of mental impairment in the community. Since goitrogenic foods were consumed during economic hardship, they were consumed more often by the poor, many of whom were native, and mental impairment may have been more common among them. In this biosocial system there were numerous ramifications into the sociocultural domain, including effects on social stratification, images of the indigene, and other ideational reactions to the high frequency of mental impairment among

the poor (Greene, 1977). In this example, there is evidence of risk allocation because risk is added through the ingestion of goitrogens. There also is evidence of biological impacts on the cultural system in that social stratification is maintained partly through the generation of relatively large numbers of cognitively impaired individuals in the population and because conceptions of the "other" are responsive to the biological features that are thought to characterize social strata and/or groups other than one's own.

The model of biocultural interaction in which culture is a risk allocator differs from previous models in several ways in addition to recognizing the possibility of mutual interactions between biology and culture. Recent interaction models based on cultural ecology and systems theory have taken culture into account as an allocator of resources for survival, health, and reproduction. Cultural contexts of social and economic inequalities structure the resources and options people draw on in responding to environmental stressors. When resources are allocated so some sectors of society are inadequately supplied, these needs are not met, and the cultural buffer wears thin for those sectors (for numerous detailed examples of such systems see Cravioto, 1970; Cravioto et al., 1967). This is seen clearly in stratified societies where, for example, nutritional resources are so unequally distributed that reproduction and survival are severely compromised in the under-allocated group. This model is basically subtractive: resources are withheld or subtracted from some sectors, leading to deficiencies that threaten health and reproduction.

While culture can diminish access to resources through socioeconomic inequalities, it can also add exposure to harmful materials or vectors of disease. These additions are modeled with the concept of risk allocation. Thus, the model of risk allocation complements the resource allocation model by recognizing the dual role that culture plays. It allocates resources and risks along lines created by social attribution. In stratified societies, these lines are created by political and economic factors and directly impact residence and occupation. Recent models of culture emanating from ethnology are consistent with this view. Culture consists of a sociopolitical context including the biological stresses of poverty to which people attempt to adapt. The risk-allocation model is related to the concept of culture that underlies political economy by emphasizing the role that residence, occupation, and class play in directing stressors in society.

In conclusion, venturing into the urban ecosystem makes observations possible that can affect our models of biocultural interaction. These effects pertain not only to pollutant exposure but to the distribution of infectious and chronic diseases as well. While culture may be seen to solve problems of health, reproduction and survival, as it does so it also creates new problems in these same domains. In the urban ecosystem, perhaps the most direct product of the human mind to influence biology, culture is both a buffer and an unequal allocator of risk.

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